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Trunk muscle coactivation is tuned to changes in task dynamics to improve responsiveness in a seated balance task

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ABSTRACT

When balancing, instability can occur when the object being balanced moves at a rate that is beyond the abilities of human motor control. This illustrates that responsiveness of motor control is limited and can be investigated by changing the dynamics of the task. In this study, the responsiveness of trunk motor control was investigated by changing the seat stiffness of an unstable seat. At decreasing levels of seat stiffness the probability of successfully balancing on the seat, speed of the seat, speed of the trunk relative to the seat (trunk-seat) and muscle activation of five trunk muscles were assessed. Also, across the different stiffness levels, the relation between trunk muscle activation and seat speed was determined. As hypothesized, with decreasing seat stiffness the probability of success decreased, seat speed and trunk-seat speed increased, and both agonist and antagonist activation increased. This shows that limits in the responsiveness of trunk motor control were reached during seated balancing. Furthermore, in line with our hypothesis, a positive relation was found between trunk muscle activation and seat speed. It appears that the central nervous system regulates trunk stiffness (via muscle coactivation) in relation to the dynamics of the task, possibly to maintain adequate responsiveness.

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1. Introduction

From systems theory, it is well known that delays in feedback control can affect the performance of a system, and if significant can lead to instability (Reeves et al., 2011). For instance, delays in motor control prevent the balancing of an upright pencil on our fingertip. Instability in balancing the pencil occurs because the dynamics of the system being controlled (the pencil) are outside the response rate of the controller (motor control system). Information processing, transmission and electromechanical delays limit the responsiveness of motor control. Quantitatively speaking, the responsiveness of control can be expressed in terms of the bandwidth, which reflects the attenuation and/or phase shift from the input (pencil starts to fall over) to the output (move fingertip under the pencil).

Using a stick balancing task, the responsiveness of human motor control was studied by lowering a mass affixed to the stick (Reeves et al., 2013). By lowering the mass, the natural frequency of stick movement increased and the task became more challenging. Eventually as the mass was lowered, limits in the responsiveness of motor control were reached, as indicated by a sharp drop-off in the probability of successfully balancing the stick. Also observed in this study was an increase in muscle coactivation in both the forearm and trunk as the mass was lowered. It was hypothesized that increased muscle coactivation was used to upregulate the responsiveness of motor control. In this way, the central nervous system (CNS) could adapt muscle coactivation to task dynamics to maintain adequate responsiveness in motor control. To clarify, agonist–antagonist coactivation does not change the net torque; however, pre-activation of agonist–antagonist muscles can decrease electromechanical delay (EMD). EMD decreases with activity level over a range of muscle activation levels from no activity to about 25% MVC, through reduced slack and increased stiffness of the muscles' series elastic components (Vint et al., 2001; Muraoka et al., 2004; Morse et al., 2005; Cavanagh and Komi, 1979). Hence, coactivation may facilitate faster responses.

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In this study, we investigated the effects of changing the dynamics of a seated balance task on trunk muscle activation. As with the stick balancing task, it was hypothesized that limits in the responsiveness of motor control would be reached as the seated balance task became more challenging, resulting in a sharp drop-off in the probability of successful balancing. It was also hypothesized that the level of trunk muscle coactivation would be linearly related to the dynamics of the task, specifically, the rotational stiffness of the seat and its speed during balancing. Although not a main focus of the study, we investigated the impact that changes in neuromuscular delays had on the probability of success for the seated balance task. A simple neuromuscular model of the seated balance task provided the opportunity to simulate effects of changes in neuromuscular delays on the probability of successful balancing.

2. Methods

2.1. Subjects

Twelve healthy subjects, six males and six females, were included in the study. The mean age, height and weight of both genders are shown in Table 1. Neurological disorders or significant visual problems interacting with balancing ability were applied as exclusion criteria. The experimental protocol was approved by the Institutional Review Board and all subjects signed informed consent prior to testing.

2.2. Experimental setup

As shown in Fig. 1, subjects were seated in an upright position with their lower legs fixed to a leg support to maintain a knee angle of approximately 90 degrees. The seat base was supported by two ball-and-socket joints in the mid-sagittal plane and by two springs in the frontal plane (see Fig. 2). The seat could only rotate in the frontal plane. The springs were fixed under tensile pre-load and had a stiffness coefficient of 4 N/cm. The task was designed to constrain the range-of-motion of the seat to ±10 degrees. Subjects were instructed to maintain the seat as level as possible during every trial. If the subject maintained the seat within the ±10 degrees range, the trial was deemed successful. If the seat exceeded the ±10 degrees range, an audible signal was triggered and the trial was deemed unsuccessful or failed.

Seated balance was assessed with six different positions of the springs supporting the seat base. The springs were positioned at equal distances with respect to the center of the seat base. The distances 2, 7, 12, 17, 22 and 27 cm were determined from pilot tests on the range of spring distances that enclosed both successful and failed balancing. These distances imposed a rotational seat stiffness of 0.3, 3.9, 11.5, 23.1, 38.6 and 58.1 Nm/rad assuming some trigonometric simplifications using maximal displacement of the seat. Ten trials were performed at every seat stiffness and every trial lasted 5 s. Two visits took place on separate days (at least 24 h and preferably 48 h in between the visits). For each visit, two sessions were performed. During a visit, the first session started at the highest stiffness level and the second session started at the lowest stiffness level.

Table 1
Means and standard deviations of age, height and weight for both genders.

	Males	Females
Age (years)	22.8 ± 3.0	22.5 ± 3.1
Height (cm)	177.7 ± 5.9	163.5 ± 7.1
Weight (kg)	78.7 ± 14.3	57.2 ± 6.8

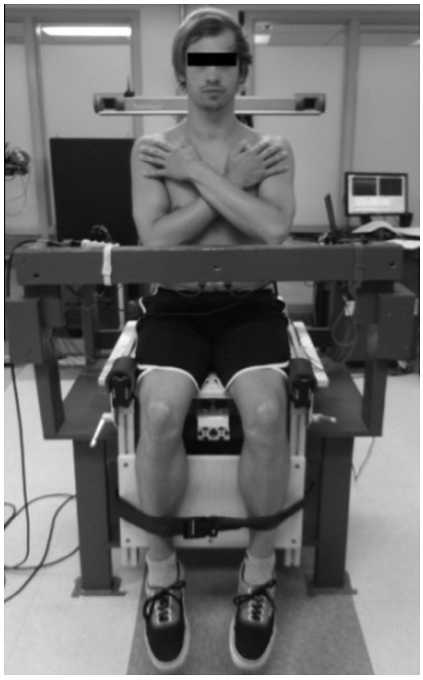

Fig. 1. Subject performing seated balance task.

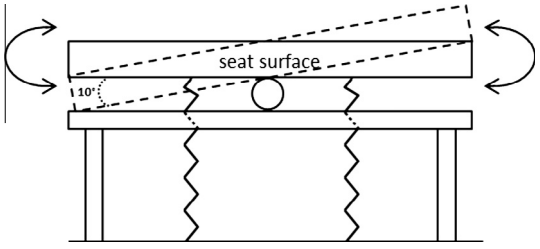

Fig. 2. Schematic representation of the seat and the support interface in a frontal plane view. The support interface consists of two ball-and-socket joints which are aligned in the mid-sagittal plane (and therefore represented by one joint in this figure) and two springs in the frontal plane. The direction of rotation is indicated by the arrows. The maximal tilt of 10° to one side is shown by the dashed seat surface.

2.3. Data collection

Seat and trunk kinematics were recorded with a motion capture system (Visualeyez Motion Capture System, Phoenix Technology Inc., Burnaby, Canada) at a sample rate of 100 samples/s. To track seat kinematics, two LED markers were fixed to the seat frame. Trunk kinematics were tracked by a LED marker attached to the skin over the spinous process of L4 and by another LED marker attached to the skin over the spinous process of T9.

Surface electromyography (EMG) signals were collected with a Delsys (Boston, USA) Bagnoli-16 Main amplifier unit (16-bit resolution, common mode rejection ratio of minimally 84 dB, channel frequency response 20–450 Hz, input impedance $>10^{15}$ Ω//0.2 pF, noise 1.2 μV, main amplifier gain 1 k–10 k, see Van Boxtel (2001), De Luca et al. (2010) for justification for the high pass filtering at 20 Hz), DE 2.1 single differential surface EMG sensors (two 10 × 1 mm silver parallel-bar contacts with a contact spacing of 10 mm), and a National Instruments (Austin, USA) BNC-6036E data acquisition unit (16-bit resolution) at a sample rate of 1600 samples/s. The EMG sensors were attached to the skin after shaving and cleaning with alcohol until slight abrasion of the skin. Muscle activity of the following muscles was recorded bilaterally:

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rectus abdominis (RA, vertical EMG sensor orientation, approximately 3 cm lateral to the umbilicus), external oblique (EO, oblique EMG sensor orientation, approximately 3 cm anterior from the mid-axillary line between the iliac crest and the tenth rib), internal oblique (IO, slight oblique EMG sensor orientation, approximately midway between the ASIS and symphysis pubis, above the inguinal ligament), thoracic erector spinae (TE, vertical EMG sensor orientation, approximately 3 cm lateral to T9 spinous process) and lumbar erector spinae (LE, vertical EMG sensor orientation, approximately 3 cm lateral to L4 spinous process). EMG and kinematic data were synchronized with a trigger system.

To normalize EMG signals, maximal voluntary contractions (MVCs) against manual resistance were executed prior to seated balancing. Before recording of MVCs, subjects were instructed how to perform the different isometric exertions. Single isometric MVCs were performed with the trunk in a neutral posture in the exertion directions of trunk flexion, extension and left and right lateral flexion. MVC trials had a duration of 5 s, with approximately 1 min between consecutive trials.

2.4. Data analysis

All data analysis was performed using custom-made scripts in Matlab R2011A. (MathWorks, Natick MA, USA).

The number of successful and failed trials for each subject at each seat stiffness was used to calculate the probability of successful balancing, similar to Reeves et al. (2013). The data were averaged over visits and sessions.

Seat and trunk kinematic data were used to determine the speed of the seat and of the trunk relative to the seat (trunk-seat). Seat and trunk angles were calculated in a global coordinate system, in the frontal plane. Seat angles were determined using the tangent rule and the trunk angles were determined using the vector formulation of the cosine rule. Subsequently, the trunk-seat angle was defined as trunk minus seat angle. The cumulative path of the seat and trunk-seat was determined by the sum of the absolute instantaneous changes in angle. This cumulative path was subsequently divided by the duration of the trial (5 s), to obtain the seat and trunk-seat speed. Speed was chosen based on its reliability in assessing postural control (Cholewicki et al., 2000; Lariviere et al., 2013). Seat and trunk-seat speed were averaged over the successful trials and subsequently over visits and sessions for every seat stiffness.

EMG signals were demeaned, full wave rectified and low pass filtered at 2 Hz using a second order dual-pass Butterworth filter to form a linear envelope. Muscle activation was converted to % MVC using the peak EMG obtained during MVCs. Subsequently, the activity between left and right sided muscles of every muscle at each time instance was compared to determine the most active side. The agonist was defined as the most active side and the antagonist muscle as the least active side. Both agonist and antagonist activity were averaged over the entire trial duration and subsequently over the number of successful trials, and over visits and sessions for each seat stiffness.

2.5. Statistics

All statistical analysis was performed with SPSS Statistics 22 (IBM Software, Armonk NY, USA).

Before statistical testing, all data were tested for normality by visual inspection of Q–Q plots and boxplots of the data within the 6 stiffness groups. Also, a Shapiro–Wilks test was run and *z*-scores of skewness and kurtosis were determined.

For the probability of success, the data appeared to violate the assumption of normality and therefore non-parametric tests were used. To test the effect of seat stiffness on probability of success a Friedman's ANOVA was conducted. Wilcoxon signed-rank tests were used to identify where specific differences occurred between adjacent stiffness levels. A Bonferroni correction was applied to correct for the number of post hoc comparisons.

Both seat and trunk-seat speed appeared to violate the assumption of normality. However, the ANOVA is robust to violations of normality and therefore these outcomes were tested with parametric tests. The effects of seat stiffness on seat and trunk-seat speed were both tested with one-way repeated measures ANOVAs. Paired *t*-tests were used to identify whether specific differences occurred between adjacent stiffness levels. A Bonferroni correction was applied to correct for the number of post hoc comparisons. The assumption of sphericity was checked according to Girden (1992). If the Greenhouse–Geisser epsilon was above 0.75, the Huynh–Feldt correction was used, otherwise the Greenhouse–Geisser correction was used.

For EMG amplitudes, the assumption of normality was confirmed and the effects of seat stiffness on agonist and antagonist activity of five trunk muscles was tested with a one-way repeated measures MANOVA for each outcome variable. As follow-up, the effect of seat stiffness on each muscle was tested with a one-way repeated measures ANOVA, and subsequently contrasts were used to test for differences between adjacent stiffness levels. The assumption of sphericity was checked according to Girden (1992). If the Greenhouse–Geisser epsilon was above 0.75, the Huynh–Feldt correction was used, otherwise the Greenhouse–Geisser correction was used.

To test if muscle activity was related to the seat speed, linear models were constructed with generalized estimating equations (GEE). To account for the correlation between repeated measures and between subject differences at baseline, difference variables were computed for muscle activity and seat speed. Difference variables were calculated between the highest seat stiffness level and the five other levels of seat stiffness. Subsequently, in the GEE models differences in muscle activation for both agonist and antagonist were related to the difference variable in seat speed. In the GEE model an autoregressive (AR(1)) working correlation matrix was used. The muscle activations predicted by the GEE models were correlated to the measured difference variables, to describe the goodness of fit of the GEE models.

2.6. Model simulations

To assess the susceptibility on the probability of success to neuromuscular delays for the seated balance task, we conducted simulations with a dynamic model of the seated balance task (see Appendix A for more details). Briefly, neuromuscular control consisted of a partial-state feedback controller with a second-order Padé approximation for neuromuscular delay and a first-order approximation for muscle dynamics. Parametric values for a single subject were fitted using experimental data generated from the seated balance task. Neuromuscular noise based on experimental data representing zero-mean Gaussian white noise was included in the model.

Using this model, 100 seated balance simulations were performed at each of 50 seat rotational stiffness levels, linearly spaced from 4 to 60 Nm/rad. Every simulation where the lower body angle did not exceed ±10 degrees within 5 s was marked as a success. The probability of success was estimated based on the outcomes of the 100 simulations.

This procedure was then repeated with a feedback delay 30% greater and 30% less than the nominal value (25.2 ms) to reflect the range of values observed with trunk muscle reflex responses, and observed differences between people with and without back pain (Radebold et al., 2000; Reeves et al., 2005).

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3. Results

In general, movement of the seat and the trunk relative to the seat increased as seat stiffness decreased. This is illustrated by a typical example shown in Fig. 3. As expected, the increase in seat and trunk speed with decreasing seat stiffness coincided with more frequent balance loss. Seat stiffness significantly affected the probability of success ($\chi^2(5) = 57.35$, $p < 0.001$), which decreased significantly between all adjacent stiffness levels as seat stiffness was reduced, except between the two lowest levels (3.9 and 0.3 Nm/rad) (Fig. 4).

Seat stiffness also had significant effects on seat speed ($F(2.76;16.55) = 32.33$, $p < 0.001$) and trunk-seat speed ($F(3.93;17.58) = 35.99$, $p < 0.001$; Fig. 5). Seat speed increased significantly between seat stiffness 58.1 and 38.6 Nm/rad. Trunk-seat speed showed similar differences with additional significant increases between 38.6 and 23.1 Nm/rad, and between 23.1 and 11.5 Nm/rad.

Seat stiffness had a significant effect on the agonistic activity of the five trunk muscles (Pillai's Trace, $F(25,150) = 1.724$, $p = 0.025$; Fig. 6). Univariate repeated measures ANOVAs for these muscles separately revealed significant increases of muscle activity with decreasing seat stiffness in EO, IO, TE and LE agonistic activity, but not in the RA agonistic activity. Seat stiffness also had a significant effect on the antagonistic activity of the five trunk muscles (Pillai's Trace, $F(25,150) = 1.67$, $p = 0.03$; Fig. 6). Univariate repeated measures ANOVAs for these muscles separately revealed significant increases of muscle activity with decreasing seat stiffness in EO, IO and TE antagonistic activity, but not in RA and LE antagonistic activity.

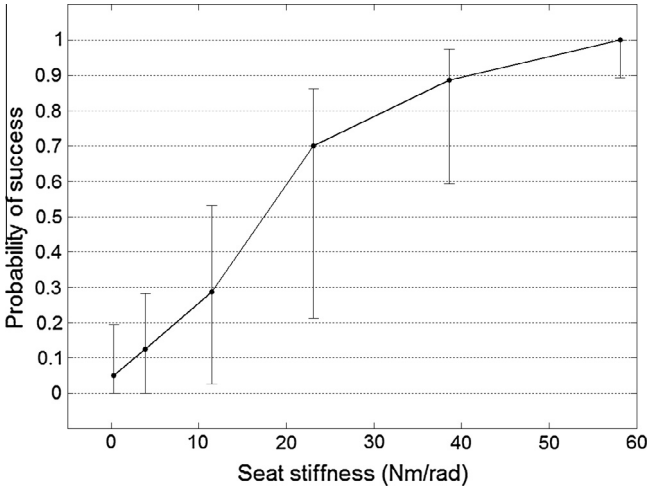

Fig. 4. Median probability of success with error bars indicating interquartile ranges at each seat stiffness.

Agonistic and antagonistic muscle activity were linearly related to seat speed for the RA, EO and IO muscles (Fig. 7 and Table 2). The TE and LE muscles showed significant linear relationships only for changes in agonistic muscle activity.

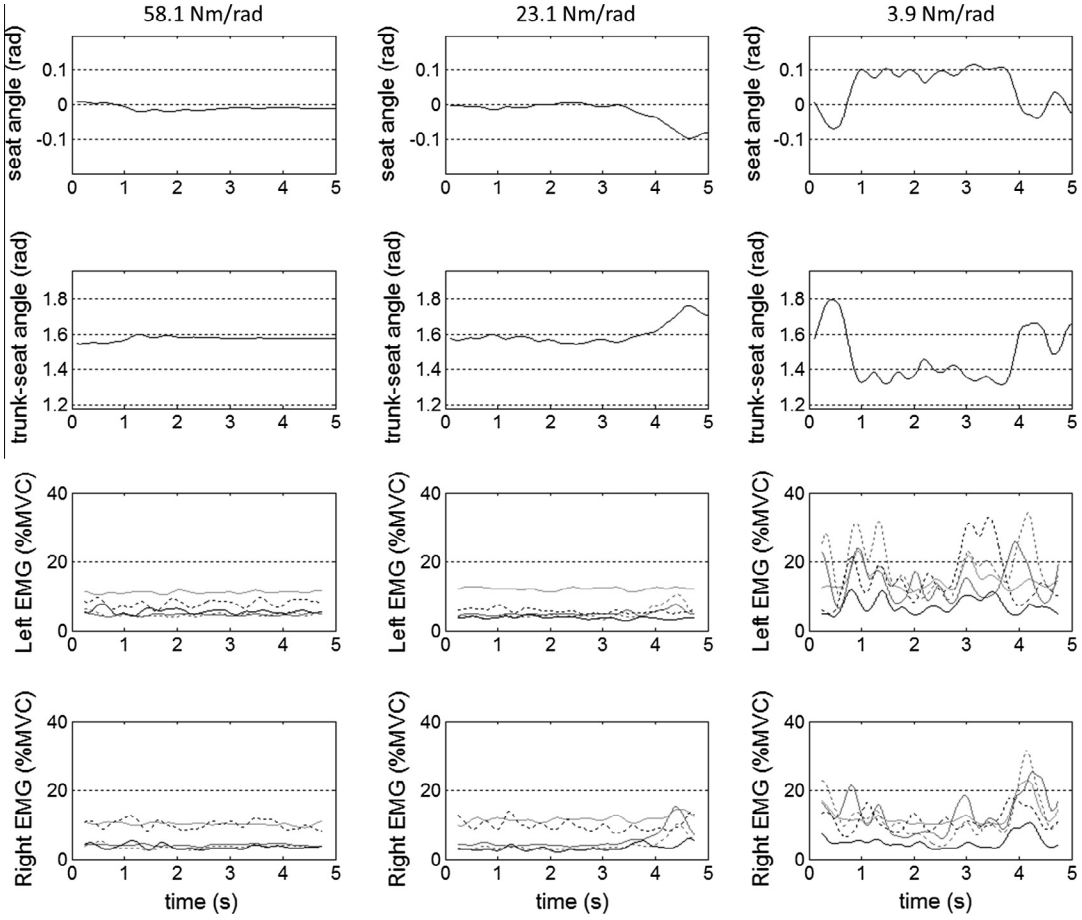

Fig. 3. Seat and trunk-seat angle time series data and EMG data of both left and right trunk muscles are shown for one subject at seat stiffness of 58.1, 23.1 and 3.9 Nm/rad. Line types of the EMG data represent the different muscle groups: solid light gray, RA; solid gray, EO; dashed gray, IO; solid black, TE; dashed black, LE.

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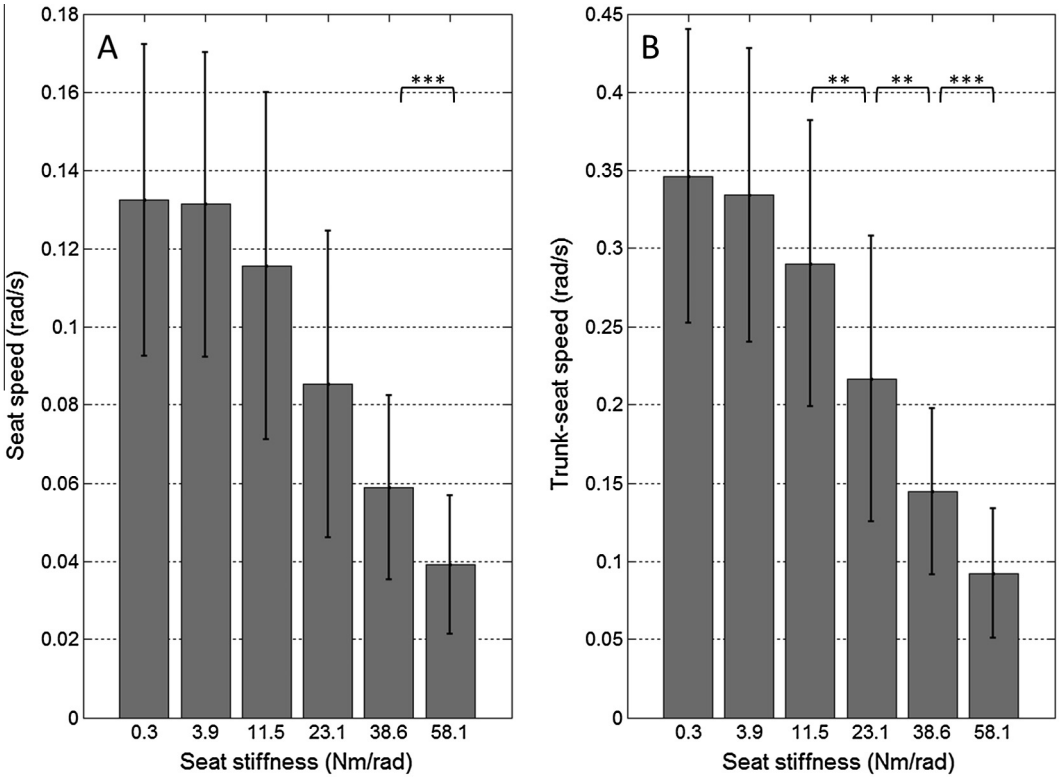

Fig. 5. Mean seat (A) and trunk-seat (B) speed with error bars indicating the standard deviations at each seat stiffness. ** represents $p \leqslant 0.01$, *** represents $p \leqslant 0.001$.

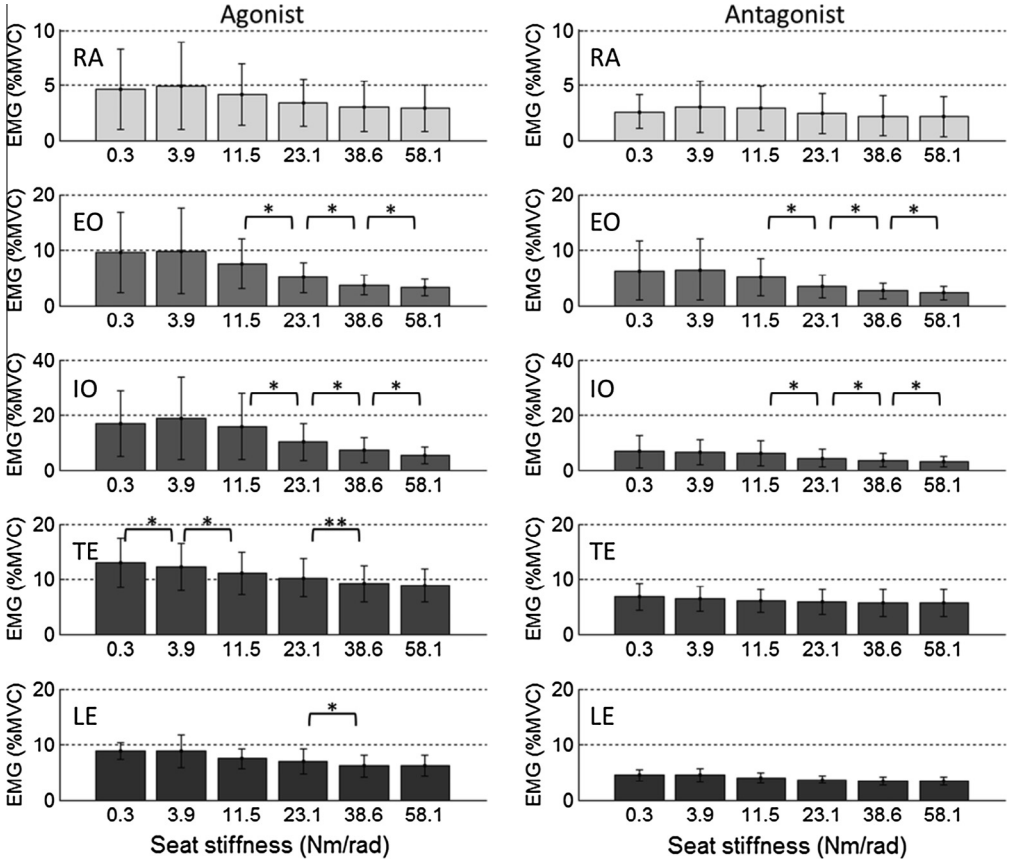

Fig. 6. Means and standard deviations of agonistic (left column) and antagonistic activity (right column) of the trunk muscles at each seat stiffness. * represents $p \leqslant 0.05$, ** represents $p \leqslant 0.01$.

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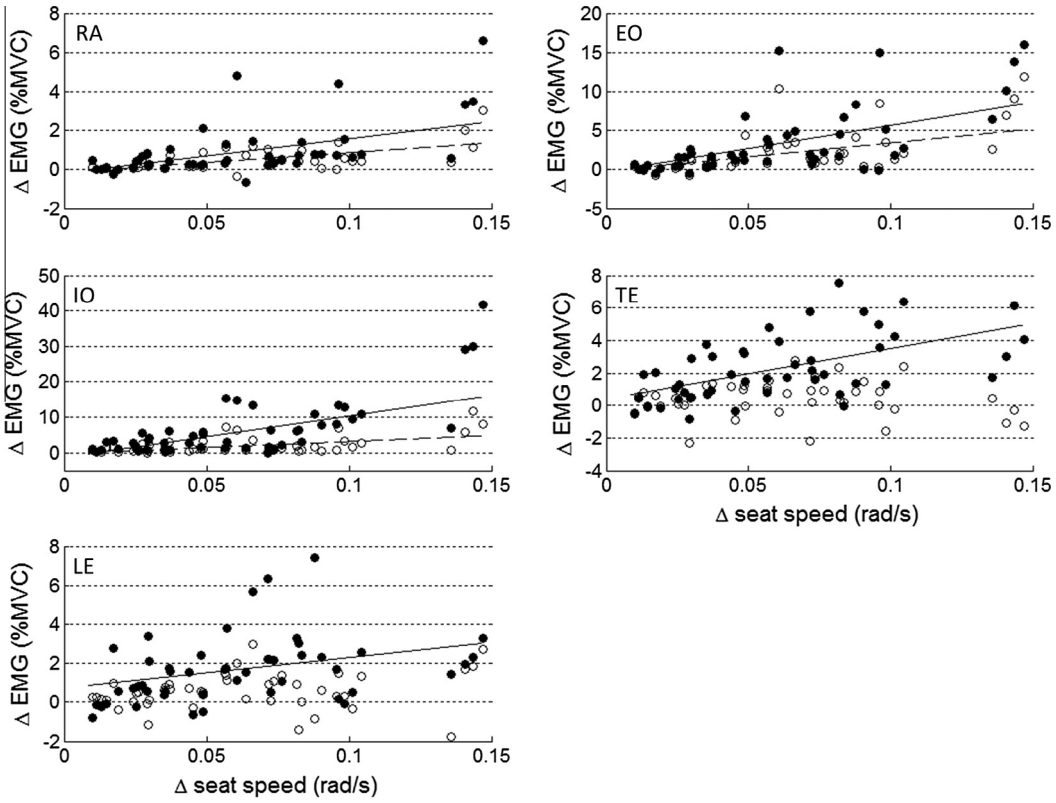

Fig. 7. Change in trunk muscle activity with respect to changes in seat speed with agonist activity in closed circles and antagonist activity in open circles. The linear regression lines for agonist and antagonist activity are shown respectively as solid and dashed lines.

Table 2
Wald χ^2 test statistic and correlation coefficients of seat speed with both agonistic and antagonistic trunk muscle activity.

Agonist			Antagonist		
	Wald $\chi^2(1)$	r		Wald $\chi^2(1)$	r
RA	10.02**	0.58***	RA	13.56***	0.61***
EO	14.03***	0.65***	EO	9.64**	0.64***
IO	8.70**	0.73***	IO	7.81**	0.55***
TE	29.41***	0.54***	TE	0.01	−0.10
LE	7.47**	0.35*	LE	2.10	0.23

* $p \leqslant 0.05$.
** $p \leqslant 0.01$.
*** $p \leqslant 0.001$.

The simulation of different neuromuscular delays showed that the probability of successfully balancing was affected by a change in delay (Fig. 8).

4. Discussion

The main goal of this study was to investigate the effect of changes in seated balance task dynamics on trunk muscle activation and more specifically to answer the question whether lower seat stiffness and higher seat speed are correlated to increased muscle activation. In general, the results suggest that the CNS is regulating trunk muscle activation to match the dynamics of the task: as seat stiffness decreased and the seat and trunk-seat speed increased, trunk muscle activation increased. Most of the changes in seat and trunk-seat speed occurred at the highest seat stiffness levels suggesting a ceiling effect. It is possible that as the seat stiffness was reduced, limits in the responsiveness of motor control were reached, which in turn resulted in a sharp drop-off in the probability of successful balancing. Therefore, the seated balance task with its range of seat stiffness appears to provide some insight into the responsiveness of the trunk.

With respect to trunk muscle activation, the data provide evidence that the CNS regulates the level of activation in relation to seat speed. This is not surprising in some sense given that the faster movements that occurred as the task became more challenging, required higher levels of agonist muscle activation to generate higher torques to change trunk angle more abruptly. But it should be noted that higher levels of antagonist muscle activation were also observed with changes in seat stiffness. In line with our

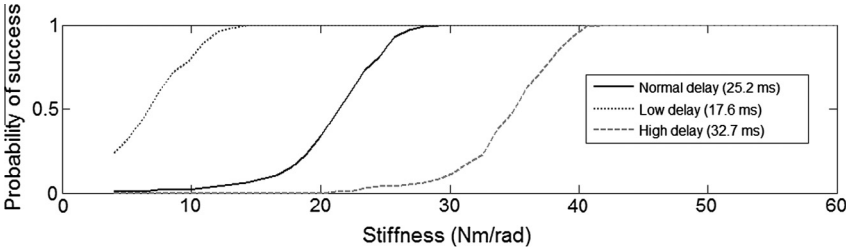

Fig. 8. Model simulations assessing the effect of changes in delays (plus/minus 30% of fitted estimate – normal) on the probability of successfully balancing.

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hypothesis, it appears that the CNS is regulating trunk stiffness in relation to seat stiffness, through agonist–antagonist muscle coactivation, in order to maintain ample control responsiveness. Pre-activation of the muscles (i.e., activation of the muscles before the targeted torque is produced), decreases the EMD (Vint et al., 2001), and therefore allows for a faster response, allowing for the stabilization of faster moving systems. To clarify, with concurrent increased right and left trunk muscles activation, when the trunk is accelerated for example from right to the left side, the pre-activated antagonists at the right side, counteract the targeted acceleration, but will become the agonists when the movement needs to be reversed. Pre-activation of the antagonist will reduce its EMD, which in turn will make the system more responsive. An alternative explanation could be that the reduction in seat stiffness needs to be compensated by an increase in trunk stiffness. Edwards (2007) showed in a model of standing balance for coupled segments that decreasing the stiffness in one joint, requires an increase of the stiffness in the other joints in order to maintain a stable static equilibrium. Since the trunk and the seat act in series in the seated balance task, it is possible that reducing the seat stiffness required increasing trunk stiffness to maintain the overall system stiffness.

The impact of neuromuscular delays on the probability of successful balancing was demonstrated with model simulations. The finding that neuromuscular delays affect postural control confirms previous unstable seated balance modeling work (Reeves et al., 2009). This previous work showed that longer delays increased the number of unsuccessful trials, the amount of trunk-seat displacement, and the trunk effort required to maintain stable balance. These effects were observed for 10–20 ms longer delays, which is close to the range in the present study (low–high difference 15 ms). There are a few discrepancies between the two modeling simulations. In the previous model simulation, the task involved balancing on a hemi-cylinder, based on the experimental set-up of Cholewicki et al. (2000) and Radebold et al. (2000, 2001), whereas the present modeling simulation involved balancing on a hinge joint, based on Slota et al. (2008), Lee and Granata (2008) and the present study. Using unstable seated balancing on a hemisphere, it was shown that compared to healthy subjects, back pain subjects with longer reflex delays (approximately 15 ms) had poor postural control and were more likely to be unsuccessful as the task became more challenging (Radebold et al., 2001). This also supports the notion that delays are linked to postural stability. And since the CNS appears to be regulating muscle agonist–antagonist activation to task dynamics, it suggests that the CNS is regulating muscle coactivation to reduce control delays (e.g. EMD).

In terms of clinical implications, now that baseline measures for a healthy population have been established, the study protocol can now be applied to populations where motor control responsiveness may be limited. Back pain and elderly populations that have been shown to have longer reflex delays are obvious candidates for this type of investigation (Radebold et al., 2000, 2001; Magnusson et al., 1996; Wilder et al., 1996; Reeves et al., 2005; Hwang et al., 2008). Furthermore, application and refinement of neuromuscular modeling could be used to give insight into the source of impairment in afflicted groups, which can then be used to guide treatment.

Conflicts of interest

The authors declare that there are no conflicts of interest.

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Appendix A

We constructed a simple model to capture the relevant dynamics of the seated balance task. The assumed structure of the model is shown in Fig. 9. In this model structure, $G(k)$ is the physical dynamics of the human/seat system, which were derived using the Lagrange equations. The controller was assumed to be a Linear Quadratic Regulator (LQR), which applies feedback based only on the 4 states of $G(k)$ (upper body angle and rate, and lower body angle and rate), and whose output is u, the human control torque applied at the L4 spinal level. The feedback loop contains added neuromuscular noise $w \sim N(0, W)$. An ideal delay τ, implemented as a 2nd-order Padé approximation, and first-order muscle dynamics with time constant T_W were also included.

The fixed physical subject parameters in $G(k)$ consisted of masses, moments of inertia, COM locations, delays, intrinsic stiffness/damping, and muscle dynamics. These parameters were taken to be those estimated from a single subject in a different study using a similar seated balance task (Priess et al., 2014). $G(k)$ is also an explicit function of the rotational seat stiffness k.

The controller K was allowed to vary based on the rotational stiffness k of the seat. Humans must adapt their own control logic to the task at hand, and a fixed controller that is stable at a high stiffness may be unstable at a low stiffness (or vice versa). To account for this, we assumed that the subject's LQR design at any stiffness level used a fixed state penalty $Q = I_4$, and an unknown fixed input penalty R. We determined R and the assumed neuromuscular noise intensity W to match the experimental probability of success at 3 different stiffness levels. At each stiffness level, an LQR control was designed using Q and a guess for R, and the closed-loop system was simulated for 5 s with added neuromuscular noise with a guess for the covariance W. A simulation was considered a "success" if the lower body angle did not exceed ±10 degrees during the simulation. R and W were then fitted using nonlinear least-squares fitting to the probability of success over 100 simulations, i.e.

$$(R^*, W^*) = \underset{R,W}{argmin} \sum_{i=1}^{3} [p_s(S|k_i) - p_e(S|k_i)]^2, \; k = \begin{Bmatrix} 39.4 \\ 23.5 \\ 4 \end{Bmatrix},$$

where $p_s(S|k_i)$ is the simulated probability of success (#successes/100) at the ith stiffness level, and $p_e(S|k_i)$ is the experimentally determined probability of the same event. The fitted values for R and W are

$$R = 0.007, W = 8 \times 10^{-4}.$$

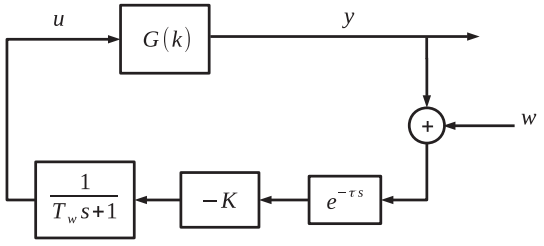

Fig. 9. Seated balance model used to investigate the effects of neuromuscular delays $e^{-\tau s}$ on the probability of successfully balancing.

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To show how the probability of success changes with stiffness, 100 5-s simulations at each of 50 stiffness levels k were performed. Each simulation used a controller designed using the fitted R, and contained added neuromuscular noise w with covariance equal to the fitted W. The stiffness levels were linearly spaced between 4 and 60 Nm/rad. A simulation was again considered a "success" if the lower body angle did not exceed ±10 degrees during the simulation. The probability of success at each stiffness level was computed as #successes/100.

To show the effect of feedback delay on the probability of success, this set of 5000 simulations was repeated with delay values that were 30% higher and 30% lower than the baseline, respectively.

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